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l4 and l5	11

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USPT	(hepatitis adj c) or hcv	1487	<u>L5</u>
USPT	thymosin same interferon	249	<u>L4</u>
USPT	thymosin.ti,ab,clm. and hepatitis	18	<u>L3</u>
USPT	thymosin.ti,ab,clm.	90	<u>L2</u>
USPT	thymosin	562	<u>L1</u>

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Terms	Documents
17 not 15	6

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PGPB,JPAB,EPAB,DWPI	17 not 15	6	L8
PGPB,JPAB,EPAB,DWPI	16 and hepatitis	13	L7
PGPB,JPAB,EPAB,DWPI	interferon same thymosin	30	L6
PGPB,JPAB,EPAB,DWPI	11 and 14	7	L5
PGPB,JPAB,EPAB,DWPI	12 and 13	30	L4
PGPB,JPAB,EPAB,DWPI	interferon	5563	L3
PGPB,JPAB,EPAB,DWPI	thymosin	226	L2
PGPB,JPAB,EPAB,DWPI	(hepatitis adj c) or hcv	1563	L1

09997758 99047262 PMID: 9831367

Interferon and thymosin combination therapy in naive patients with chronic hepatitis C: preliminary results.

Moscarella S; Buzzelli G; Romanelli RG; Monti M; Giannini C; Careccia G; Marrocchi EM; Zignego AL

Istituto di Medicina Interna, Universita' di Firenze, Florence, Italy.

Liver (DENMARK) Oct 1998, 18 (5) p366-9, ISSN 0106-9543

Journal Code: L74

Languages: ENGLISH

Document type: Clinical Trial; Journal Article; Randomized Controlled Trial

Record type: Completed

Subfile: INDEX MEDICUS

AIMS/BACKGROUND: This randomized study was performed to compare the efficacy of interferon-alpha (IFN-alpha) + thymosin alpha 1 (Talphal) treatment to that of IFN-alpha alone in light of biochemical and virological response of naive patients with chronic hepatitis C. METHODS: Seventeen patients were treated with IFN alpha-2b (3 million units MU three times a week) + Talphal (1 mg twice weekly); the other 17 patients received only IFN alpha-2b at the same dose. All patients were treated for 6 months and followed up for 12 months. Biochemical (ALT values) and virological (HCV-RNA) responses to treatment were determined. RESULTS: Combination therapy showed significantly higher efficacy than monotherapy in achieving biochemical and virologic end-of-treatment response ($p < 0.05$). At 12 month follow-up, the sustained biochemical response was slightly greater in patients treated with combination therapy than in those treated with monotherapy. No significant difference in response by HCV-1b subtype was observed between the two treatment groups; however, HCV-2c subtype showed a trend to responding better to IFN-alpha+Talphal than to IFN-alpha alone. CONCLUSIONS: These data suggest that the immune modulator Talphal may be additive or synergistic with IFN-alpha in normalizing end-treatment biochemical and virological responses in patients with chronic hepatitis C. Higher doses and/or more prolonged courses may improve the sustained response rates to this treatment.

Tag

09/544,108

Chronic hepatitis B and C. What is the status of drug therapy?

Wright TL

Division of Gastroenterology and Hepatology, School of Medicine,
University of California, San Francisco.

Postgraduate medicine (UNITED STATES) Sep 15 1992, 92 (4) p75-82,
ISSN 0032-5481 Journal Code: PFK

Languages: ENGLISH

Document type: Clinical Trial; Journal Article; Review; Review, Tutorial

Record type: Completed

Subfile: AIM; INDEX MEDICUS

Chronic hepatitis remains difficult to treat. Use of interferon has been successful against both hepatitis B and C viruses, but the outcome of long-term administration has yet to be determined. Not all patients respond to interferon, however, and some have side effects that cause them to discontinue therapy. Dr Wright discusses the results of studies to evaluate therapy with alpha, beta, and gamma interferon as well as with other agents, such as ribavirin, thymosin, and ursodeoxycholic acid. (32 Refs.)

Tags: Human

Descriptors: *Hepatitis B--therapy--TH; *Hepatitis C--therapy--TH;
*Interferons--therapeutic use--TU; Chronic Disease; Drug Costs; Interferon
Alfa-2b--therapeutic use--TU; Interferons--contraindications--CT; Interfer-
ons--pharmacology--PD

CAS Registry No.: 9008-11-1 (Interferons); 99210-65-8 (Interferon
Alfa-2b)

Prospectives on the treatment of chronic hepatitis B and chronic hepatitis C with thymic peptides and antiviral agents.

Mutchnick MG; Ehrinpreis MN; Kinzie JL; Peleman RR
Department of Medicine, Wayne State University School of Medicine,
Detroit, MI 48201.

Antiviral research (NETHERLANDS) Jul 1994, 24 (2-3) p245-57, ISSN
0166-3542 Journal Code: 6I7

Languages: ENGLISH

Document type: Journal Article; Review; Review, Tutorial

Record type: Completed

Subfile: INDEX MEDICUS

At the present time, interferon is considered the only effective therapeutic approach in the treatment of both chronic hepatitis B and chronic hepatitis C. It is clear that the disappointing response rates in both chronic hepatitis B and C place added emphasis on efforts to identify alternative forms of therapy. In addition to the development of other antiviral agents including the nucleoside analogs which might prove more effective and have fewer associated side-effects, other agents currently under investigation include thymic peptides such as thymosin alpha 1. In the future, the therapeutic approach to the treatment of chronic hepatitis B and C may consist of combination therapy using perhaps an immune modulator and an antiviral agent or, several antiviral drugs. Alternatively, there is indication that cellular targeting systems with delivery of the toxic material to the specific cell containing the virus may be more effective, while minimizing side-effects. Finally, there are agents such as ursodeoxycholic acid which perhaps, makes bile less toxic and can be used as adjunctive therapy with improvement in liver chemistry values. The treatment of chronic hepatitis B and chronic hepatitis C has shifted in emphasis from the concept of treating liver disease towards that of treating viral infections which happen to effect primarily the liver. (65 Refs.)

Tags: Human

Descriptors: *Antiviral Agents--therapeutic use--TU; *Hepatitis B--drug therapy--DT; *Hepatitis C--drug therapy--DT; Adjuvants, Immunologic --therapeutic use--TU; Bile Acids and Salts--therapeutic use--TU; Chronic Disease--drug therapy--DT; Interferons--therapeutic use--TU; Nucleosides --therape

5/9/4
DIALOG(R) File 155:MEDLINE(R)

10183856 99293035 PMID: 10362814

Histological damage in chronic hepatitis C is not related to the extent of infection in the liver.

Rodríguez-Inigo E; Bartolome J; de Lucas S; Manzarbeitia F; Pardo M; Arocena C; Gosálvez J; Oliva H; Carreno V

Department of Hepatology, Fundación Jiménez Díaz and Fundación Estudio Hepatitis Virales, Madrid, Spain.

American journal of pathology (UNITED STATES) Jun 1999, 154 (6) p1877-81, ISSN 0002-9440 Journal Code: 3RS

Languages: ENGLISH

Document type: Journal Article

Record type: Completed

Subfile: AIM; INDEX MEDICUS

It has not been completely elucidated whether the liver injury induced by the hepatitis C virus (HCV) is due to direct cytopathic damage or to an immune-mediated response against HCV-infected hepatocytes. In this work, we have determined the percentage of HCV-infected hepatocytes, the histological activity index, and the viremia levels in chronically HCV-infected patients with different grades of liver injury to investigate any possible correlation between them. For that purpose, liver biopsies from 27 patients with HCV chronic hepatitis were analyzed by in situ hybridization. This technique revealed that the percentage of infected hepatocytes ranged from 0.04% to 83.6%. Regarding the viremia levels, HCV RNA concentration ranged from 1.8×10^3 to 1.4×10^6 genome copies/ml. A significant correlation ($r = 0.54$; $P = 0.003$) between the percentage of infected hepatocytes and the viremia levels was found. In contrast, no correlation was observed between the percentage of HCV-infected hepatocytes or the viremia levels and the histological activity index. In conclusion, we have shown that the HCV viremia reflects the extent of the infection in the liver and that the liver injury in chronic HCV infection is not directly related to either the number of infected hepatocytes or the serum HCV RNA concentration.

Tags: Female; Human; Male; Support, Non-U.S. Gov't

Descriptors: *Hepatitis C, Chronic--pathology--PA; *Hepatitis C, Chronic--virology--VI; *Hepatitis C-Like Viruses--pathogenicity--PY; Biopsy; DNA, Viral--metabolism--ME; Genotype; Hepatitis C-Like Viruses--isolation and purification--IP; In Situ Hybridization; RNA, Viral--blood--BL; Regression Analysis; Reverse Transcriptase Polymerase Chain Reaction; Viral Load

CAS Registry No.: 0 (DNA, Viral); 0 (RNA, Viral)

Record Date Created: 19990714

5/9/6
DIALOG(R) File 155:MEDLINE(R)

09412742 97475315 PMID: 9334839

Inflammatory markers in chronic hepatitis C.

Banner BF; Allan C; Savas L; Baker S; Barnard G; Bonkovsky HL

Department of Pathology, University of Massachusetts Medical Center, Worcester 01655, USA.

Virchows Archiv (GERMANY) Sep 1997, 431 (3) p181-7, ISSN 0945-6317
Journal Code: BZD

Contract/Grant No.: DK38825, DK, NIDDK

Languages: ENGLISH

Document type: Journal Article

Record type: Completed
Subfile: INDEX MEDICUS

To test the hypothesis that inflammation in hepatitis C follows mechanisms common to immune-activated pathways, the distributions of T and B cells, adhesion molecules and transforming growth factor-beta (TGF-beta) were assessed in liver biopsies with chronic inflammation due to hepatitis C (HCV, n = 8) and other causes (non-HCV, n = 10). Frozen sections were immunostained using primary antibodies to CD2, CD20, CD4, CD8, intercellular adhesion molecule (ICAM-1), vascular cell adhesion molecule (VCAM)-1, HLA-DR, lymphocyte function-associated antigen (LFA)-1, and TGF-beta. Inflammatory cells positive for each immunophenotypic marker were counted, and positive staining for adhesion molecules, HLA-DR and TGF beta was graded in triads and lobules and compared in HCV and non-HCV biopsies. In all biopsies, T cells were more frequent than B cells, both in triads and lobules. CD20+, CD4+, CD8+ and LFA-1+ cells were increased in HCV compared to non-HCV biopsies. Portal lymphoid aggregates were present in 6 of 8 HCV biopsies and 3 of 10 non-HCV biopsies. Aggregates consisted of CD20+, CD4+, CD8+ and LFA-1+ cells, and ICAM-1 and VCAM-1 were increased. Sinusoidal lining cells in HCV biopsies and non-HCV biopsies with inflammation expressed HLA-DR, ICAM-1, and CD4. TGF-beta was increased in foci of necrosis. Inflammation in chronic HCV involves common immune-mediated cellular effector pathways and the inflammation in the portal triads represents aggregation of both T and B cells, mediated in part by upregulation of adhesion molecules on portal stromal cells; this is possibly in response to antigens draining from necroinflammatory foci in the lobules. TGF-beta is increased in active necroinflammatory foci, but not in portal lymphoid aggregates.

Tags: Comparative Study; Female; Human; Male; Support, Non-U.S. Gov't; Support, U.S. Gov't, P.H.S.

Descriptors: *Biological Markers--analysis--AN; *Hepatitis C, Chronic--immunology--IM; *Liver Diseases--immunology--IM; Adult; Aged; Antigens, CD--analysis--AN; Biopsy; Hepatitis C, Chronic--pathology--PA; Immunohistochemistry; Intercellular Adhesion Molecule-1--analysis--AN; Liver Diseases--pathology--PA; Lymphocyte Function-Associated Antigen-1--analysis--AN; Middle Age; Transforming Growth Factor beta--analysis--AN; Vascular Cell Adhesion Molecule-1--analysis--AN

CAS Registry No.: 0 (Antigens, CD); 0 (Biological Markers); 0 (Lymphocyte Function-Associated Antigen-1); 0 (Transforming Growth Factor beta); 0 (Vascular Cell Adhesion Molecule-1); 126547-89-5 (Intercellular Adhesion Molecule-1)

Record Date Created: 19971103

5/9/11
DIALOG(R) File 155:MEDLINE(R)

09003581 96384177 PMID: 8792074

Hepatocellular injury in hepatitis B and C virus infections.

Feitelson MA

Department of Pathology, Anatomy, and Cell Biology, Thomas Jefferson University, Philadelphia, Pennsylvania, USA.

Clinics in laboratory medicine (UNITED STATES) Jun 1996, 16 (2)
p307-24, ISSN 0272-2712 Journal Code: DLS

Contract/Grant No.: CA48656, CA, NCI; CA66971, CA, NCI

Languages: ENGLISH

Document type: Journal Article; Review; Review, Academic

Record type: Completed

Subfile: INDEX MEDICUS

Most of the liver cell injury in hepatitis B and C infections is likely to be immune-mediated. Variation in the pathogenesis of these infections likely is contributed by a variety of host and virus factors. Host factors include the human leukocyte antigen (HLA) haplotype as well as the ability of the host to both recognize antigen on virus-infected cells and to receive the appropriate co-stimulatory signals in a timely fashion during infection. Virus factors include the genetic variation, direct cytopathic

effects, and the alteration of infected hepatocytes to cytotoxic cytokines. The lack of suitable tissue culture systems and animal models limits the ability to understand the pathogenesis fully but provides challenges for their future development so that the basis for liver cell damage can be elucidated and approaches for therapeutic intervention can be achieved. (110 Refs.)

Tags: Animal; Human; Support, U.S. Gov't, P.H.S.

Descriptors: *Hepatitis B--pathology--PA; *Hepatitis C--pathology--PA; *Liver--pathology--PA; Hepatitis Antigens--immunology--IM; Hepatitis B--immunology--IM; Hepatitis C--immunology--IM; Histocompatibility Antigens Class I; Interferons; Liver--immunology--IM

CAS Registry No.: 0 (Hepatitis Antigens); 0 (Histocompatibility Antigens Class I); 9008-11-1 (Interferons)

Record Date Created: 19961101

5/9/16

DIALOG(R) File 155:MEDLINE(R)

08384623 95113671 PMID: 7529220

Pathology of hepatitis C.

Uchida T

Department of Pathology, Nihon University School of Medicine, Tokyo, Japan.

Intervirolgy (SWITZERLAND) 1994, 37 (2) p126-32, ISSN 0300-5526
Journal Code: GW7

Languages: ENGLISH

Document type: Journal Article; Review; Review, Tutorial

Record type: Completed

Subfile: INDEX MEDICUS

Damage and necrosis of hepatocytes in viral hepatitis C is considered to be immune mediated as in hepatitis B. Hepatocellular necrosis accompanies infiltration of lymphocytes and this feature, called necroinflammation, characterizes all types of viral hepatitis and corresponds to the histological expression of hepatitis. Although the histological features of hepatitis C do not differ fundamentally from those of hepatitis B, there are some quantitative differences. Weak but constant necroinflammation and a strong lymphocytic reaction of the portal tracts appear to be relatively unique to chronic hepatitis C. Nearly all chronic hepatitis C cases do not improve during the natural course of infection; however, in a limited number of cases, interferon treatment can eliminate the virus leading to normalization. The pattern and extent of fibrosis can roughly predict the efficacy of interferon treatment. (10 Refs.)

Tags: Human

Descriptors: *Hepatitis C--pathology--PA; Antigens, Viral--analysis--AN; Hepatitis C--immunology--IM; Hepatitis C--therapy--TH; Hepatitis C Antigens; Interferons--therapeutic use--TU

CAS Registry No.: 0 (Antigens, Viral); 0 (Hepatitis C Antigens); 9008-11-1 (Interferons)

Record Date Created: 19950209